portion of the splint, sometimes fails to maintain reduction; and the use of weight extension for fractures of the upper extremity, confining the patient to bed, is unsatisfactory in the extreme.

SPLENIC ANÆMIA, WITH HÆMOGLOBINÆMIA AND DECREASING SPLENOMEGALY.

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OSLER[1] describes splenic anæmia as "a chronic affection, probably an intoxication of unknown origin, characterized by a progressive enlargement of the spleen, which cannot be correlated with any known cause, as malaria, leukæmia, syphilis, cirrhosis of the liver, etc. (primary splenomegaly); anæmia of a secondary or chlorotic type (leukopenia); a marked tendency to hemorrhage, particularly from the stomach; and in many cases a terminal stage, with cirrhosis of the liver, jaundice, and ascites (Banti's disease)."

Banti's[2] definition is that the disease is "an idiopathic progressive anæmia without leukæmia, accompanied by idiopathic splenic hypertrophy, sometimes also with hepatic hypertrophy."

These definitions, however, do not set the subject at rest. There is still some doubt or denial that splenic anæmia is an independent disease; but the tendency is toward admitting its individuality, yet those that admit the individuality not seldom confuse the subspecies. Osler[3] is of the opinion that "a special malady does exist," and he thinks that primitive splenomegaly, splenic anæmia, splenomegalic cirrhosis of the liver, or Banti's disease, are stages of one disease.

Herman Senator, at the Seventy-first annual meeting of the British Medical Association, July, 1903,[4] said that "splenic anæmia as a special disease cannot be distinctly differentiated from similar affections of the hæmopoietic viscera, which are called pseudoleukæmic." This opinion has still considerable following in Germany, but is almost abandoned here. Senator is also of the opinion that the liver cirrhosis in splenic anæmia is very probably secondary, and a consequence of the splenic affection.

[1] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1900, vol. cxiv.

[2] Dell' anemia splenica, Florence, 1882.

[3] Loc. cit., November, 1902.

[4] British Medical Journal, 1903, vol. ii. p. 573.

The chief characteristic of the malady is a marked enlargement of the spleen: only in malaria, syphilis, and leukæmia is the spleen found as large as it becomes in this disease. Leukæmia may be differentiated from splenic anæmia by a blood examination. Even in that phase of leukæmia sometimes observed, wherein the number of leukocytes diminishes to near the normal counting, the myelocytes remain. In malarial cachexia it is not uncommon to find a spleen that weighs from seven to ten pounds, and there has been a case reported where it weighed more than twenty pounds; on the other hand, a malarial spleen may atrophy. Russell[1] reported a case where the spleen weighed only one ounce and nineteen grains. There are many pathological characteristics in malarial spleens in chronic cases that are similar to conditions observed in the spleen of splenic anæmia, but the pigmentation of malaria is much more marked. The trabeculæ and the areas in the neighborhood of vessels are very melanotic in protracted malaria; and the plasmodia, broken down, with pigment and the remains of cells, are often found in the splenic veins. The malarial parasite commonly disappears a short time after death, and this absence, with the fact that a malarial cachexia is possible without any previous fever, might make the diagnosis somewhat obscure. The differential blood count alone between malarial cachexia and splenic anæmia shows nothing, except that the large mononuclear leukocytes will be more numerous in malaria than in splenic anæmia. It is possible also to have a coincidence of malaria and splenic anæmia—Osler reported a number of such cases—but this is a mere coincidence and nothing more.

Syphilitic gummata may make the spleen very big, especially in congenital syphilis, but the history, the irregularity of the liver, and the more moderate enlargement of the spleen would render the diagnosis clear. The amyloid spleen of syphilis, bone caries, and other diseases have characteristic accompanying symptoms.

The microscopic appearances of the blood in splenic anæmia and pernicious anæmia occasionally have resemblance, but in pernicious anæmia the spleen is either normal in size, or reduced, or only moderately enlarged; it has been found as heavy as nineteen ounces,[2] but this size is altogether exceptional. In 40 cases of pernicious anæmia in Osler's wards, the spleen was palpable in only 6 cases—merely felt and not at all markedly enlarged. In 12 cases of splenic anæmia collected by Rolleston, the average weight of the spleen was 61 ounces; in Bovaird's case it weighed 12½ pounds, and a weight of, at the least, 30 ounces is quite common.

In primary cirrhosis of the liver with secondary splenic enlargement, the spleen does not approach the weight found in splenic anæmia. Kelynack[1] found in 84 cases of cirrhosis of the liver that the spleen weighed on an average 12.93 ounces. In another series of 114 cases[2] the average weight was 9.8 ounces—about two ounces above the normal weight. In still another series of 47 directly fatal cases of primary cirrhosis of the liver the average weight of the spleen was 11 ounces, and in 67 cases, where death came from intercurrent causes, the weight was only 9 ounces.[3] There is, moreover, an explanatory history of alcoholism or syphilis in the vast majority of the cases of primary liver cirrhosis, but this etiology is absent in splenic anæmia. Specific fevers, malaria, gout, and rachitis cause cirrhosis of the liver—some certainly, others very probably. Hamilton[4] reported a case of cirrhosis of the liver in a child, aged six years, where there was no discoverable history of syphilis or alcoholism; and Rolleston and Haight[5] described a congenital case with syphilis excluded. These very exceptional cases do not materially affect the differential diagnosis.

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[1] Malaria and Injuries of the Spleen, Calcutta, 1880.

[2] Hunter. Lancet, September 22, 1888.

Banti's disease, properly so called, is primitive splenomegaly with secondary hepatic cirrhosis, and this is at times deemed a disease distinct from splenic anæmia. Banti contends that the condition of the liver is a consequence of the splenic condition, not a cause.[6] Dock and Warthin[7] even hold they could demonstrate in one of their cases that the splenic hyperplasia was older than that in the liver, because in the spleen the fibrous tissue was relatively in greater abundance, more dense, more hyaline, different in reaction to Van Gieson's stain, and more atrophic in the parenchyma. Banti, Bovaird, Stengel, and Brill have undoubtedly found clinically in closely observed cases the splenomegaly to precede the enlargement of the liver.

There is, then, either a distinct disease, splenic anæmia with or without hepatic cirrhosis, or a new kind of cirrhosis of the liver, wherein the spleen is twice or thrice as large as the spleen in the known cases of primary hepatic cirrhosis and the usual etiology of this cirrhosis is missing, or another unnamed blood disease, differing in a marked degree from the anæmias and leukæmias already classified; in any case the condition should have a special name, and, for want of a better, we may call it splenic anæmia. Cabot reasonably objects to this name, but it is becoming as fixed as terms like hysteria and hydrophobia.

Banti, in all his articles on this disease, says the etiology is unknown, and the many theories offered to explain its origin are not convincing. Barr[8] traces the disease to a vasomotor paresis of the splanchnic area, which arises from a disease of the visceral sympathetic ganglia. The result, he says, is an engorgement of the abdominal viscera, especially the liver and the spleen, with hæmolysis as a consequence. The blood stasis leads to fibrosis and lessened functional power; it also causes the peritoneal effusion, which is, in this disease, not dependent upon portal obstruction. The same vasomotor paresis which lowers blood pressure leads to a retention of blood in the portal circulation, and consequent hemorrhage and impaired digestion. If this theory holds, the disease of the visceral sympathetic ganglia might be caused by a toxin of intestinal origin. Senator, Rolleston, and J. Mitchell Clarke are of the opinion that the toxin is of intestinal origin. That a toxin comes from the intestines is mere theory, and such intoxication has been set up as the source of as much evil as eye-strain. Sir Andrew Clark and others made it the cause of chlorosis; Bradshaw, of anæmia; Potain, of hepatic cirrhosis; Rolleston and others, of splenic anæmia; Rivière, of anæmia splenica infantum; others, of tetany, and so on almost indefinitely.

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[1] Allbutt's System of Medicine, vol. iv. p. 531.

[2] Rolleston and Fenton. Birmingham Medical Review, October, 1896.

[3] Allbutt, loc. cit.

[4] International Clinics, series 13, vol. ii. p. 245.

[5] British Medical Journal, March 30, 1901.

[6] Dell' anemia splenica, p. 26.

[7] THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, January, 1904.

[8] Lancet, August 23, 1902.

Banti thinks that the source of the splenic anæmia is the spleen itself, but the nature of this influence is unknown to him. As the normal physiology of the spleen is unsettled, it is impossible fully to determine its bearing upon any pathological condition. Bruhl[1] thinks that the atrophy of the spleen and its loss of function bring about alteration in the chemical constitution of the blood, and a consequent anæmia. If this were true, splenectomy should cause the symptoms of splenic anæmia, but it does not.

That splenectomy has improved or even cured cases of splenic anæmia does not necessarily prove that the spleen itself is the cause of the disease. Bottazzi[2] seems to find that normally the spleen, besides destroying erythrocytes takes out hæmoglobin from these corpuscles, and that removal of the spleen in dogs makes the red corpuscles less liable for a long time to lose hæmoglobin. Hunter[3] found that in rabbits after splenectomy toluylendiamin will cause only slight or no hæmolysis. If splenic anæmia is a chronic toxæmia, splenectomy may render the erythrocytes less liable to destruction by leaving the hæmoglobin more stable. This explanation for the effect of splenectomy is offered by Rolleston.[4]

Again, splenic anæmia seems not to be caused by an increase in the hæmolytic function of the spleen, because its abnormal enlargement in that malady is not a real hypertrophy, but an effect of cirrhotic atrophy; and there is not an unusual evidence of iron or pigment to show excessive destruction of erythrocytes in the spleen itself. Still, it might send out a hæmolytic enzyme to act in the general circulation—but that is a mere guess.

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[1] Archives générales de méd., June and August, 1891.

[2] Archiv. ital. di biologia, 1895.

[3] Lancet, 1892, vol. ii. p. 1259.

[4] Allbutt's System of Medicine, vol. iv. p. 523.

All the positive work done toward determining the nature of splenic anæmia has been directed toward the pulling down of explanatory theories, and we know almost nothing of the fundamental causes of the disease. The origin is probably a toxin of such a nature that it is made virulent by the physiological action of the spleen itself, and the blood changes are thus effected. The splenomegaly and the hepatic condition might be explained by congestion and loss of function, if we could find the cause of such stasis; to attribute it to vasomotor paresis is sheer conjecture. There is a condition commonly classed with splenic anæmia, in which the proliferation of endothelium is so marked as to suggest a primary endothelioma of the spleen; here again we are at a loss for causes.

The principal changes in the spleen in splenic anæmia are:

1. An atrophy of the Malpighian bodies, which is caused by an overgrowth of connective tissue about the central artery, or by an ingrowth of fibrous tissue from the periphery.
2. A thickening of the capsule, the trabeculæ, the walls of vessels, and of the reticular lining of the spaces in the splenic pulp.
3. A proliferation of the endothelial lining of the splenic vessels and the blood spaces of the pulp. This proliferating endothelium is large, with clear cytoplasm, and the nucleus is peripherally placed. The proliferation may be absent, but often it is the most noteworthy quality of the case. Based on the supposition that the dissolution of blood corpuscles normally takes place in the spleen by means of an enzyme formed from the endothelial cells of the pulp, there is an opinion that in splenic anæmia the marked increase of endothelium is the cause of the hæmolysis in that disease. The origin of the endothelial proliferation is not known, and, as has been said, in numerous cases it is not enough in evidence to explain the conditions.

Brill, in an article entitled "Primary Splenomegaly—Gaucher Type,"[1] which is a report on one of four cases occurring in a single generation of one family, and presented in collaboration with Mandelbaum and Libman, holds that primary splenomegaly is a distinct disease not to be classed with splenic anæmia. In this type, he says, the liver may be enormously enlarged as the spleen is, and the proliferation of endothelium is very marked. In his case the spleen weighed eleven pounds and the liver ten pounds—the weight of the normal spleen is very variable, but it is about seven ounces; that of the normal liver is about three pounds.

In the microscopic examination of the spleen in Brill's case the usual fibrosis was found, but added thereto was an enormous proliferation of endothelium in the splenic alveoli. These cells were found also in the liver—some in the lobules, but most in the connective-tissue spaces. The mesenteric, bronchial, and retroperitoneal lymph nodes were practically transformed into masses of endothelium, and such cells were found in the bone-marrow. Gaucher[1] would have this condition considered endothelioma of the spleen.

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[1] The American Journal of the Medical Sciences, March, 1905.

Stengel[2] makes primitive splenomegaly a division of splenic anæmia, and he thinks that while it is not possible perhaps to distinguish this variety clinically, there is a sharp distinction in the pathological characteristics. In one of his cases the presence of endothelial proliferation throughout the spleen was as marked as in Brill's case, but as the patient recovered after splenectomy we do not know whether the endothelial cells had invaded other organs. He discusses the question whether this endothelial growth is a neoplasm or a hyperplasia. In some cases the predominating characteristic is a fibrous hyperplasia accompanied with endothelial proliferation, while others, like his own third case, and we may add that of Brill's, have the appearance of a tumor with active neoplastic formation, essentially and primarily involving the endothelial cells.

Brill's case certainly looks like an endothelioma with metastases; as Stengel's case recovered after splenectomy, there were probably no metastases, if it were indeed an endothelioma; and as an endothelioma is not so malignant as other tumors of the sarcomatous group, that might in part account for the lack of metastases. There probably was none, because his case closely resembles our own. In our case we found very marked endothelial proliferation in the spleen everywhere, which was practically identical with Stengel's case (on comparison with a section from his case in the possession of Dr. A. O. J. Kelly), but in our case there was no metastasis. Brill, as we said, is of the opinion that in a genuine endothelioma of the spleen the liver should be greatly enlarged; in our case the liver was atrophic; it was, therefore, either not an endothelioma, or, if it was, the hepatic enlargement is not essential to the diagnosis. Probably the same is to be said of Stengel's third case, which, as far as we know, showed no signs of extraordinary liver enlargement.

In the discussion of Stengel's paper Osler drew attention to the importance of looking in the blood in splenomegaly for the protozoon of Donovan, especially in the cases that come in from the tropics. These organisms are oval, 3 to 6μ in diameter, each of which has two nuclei. It finally develops into an elongated pointed protozoon with a flagellum—the round and the rod-shape nuclei remain. In the early form the larger nucleus is oval and eccentrically placed, and the smaller nucleus is rod-shaped, and set perpendicularly or tangentially to the larger one. This plasmodium takes the Romanowsky stain or its modifications, and the cytoplasm then is reddish.

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[1] Splénomégalie primitive-endothelioma primitif de la rate, 1882.

[2] Transactions of the Association of American Physicians, 1904, vol. xix. p. 174.

The bodies occur singly or in pairs, but they may be in groups as if they had been embedded in a macrophage. They are found free and in phagocytes in the spleen, the liver, the mesenteric glands, the bone-marrow, the kidneys and adrenals, and in intestinal, skin, and mucous ulcers, but they are not found in the peripheral blood. They cause symptoms resembling malaria, but they are not removed by quinine. They commonly reduce the number of erythrocytes to from two to three million, and bring about leukopenia. Like malaria they increase the number of large mononuclear leukocytes. In ten of Donovan's cases the average percentage of mononuclears was 21.58—they varied from 6 to 48 per cent.

Clinically, splenic anæmia occurs four times oftener in males than in females, and it is commonly a disease of adults. The condition may be roughly divided into three stages: First, asthenia and perisplenitis; second, anæmia and splenomegaly; third, cachexia. In the third stage there may be ascites. The chief symptoms in general are splenomegaly; a progressive secondary anæmia with leukopenia; absence of external glandular swelling (the abdominal glands are commonly found somewhat enlarged post-mortem); notable chronicity, and in some cases a lasting hæmatemesis.

In the early stage asthenia is the chief symptom, with some wasting of the muscles, but the fat commonly remains. There may be pallor, a pigmentation of the skin, dyspnœa, palpitation; commonly there is no anorexia. The first symptom noticed in other cases is pain in the region of the spleen. This pain is increased on pressure, it radiates toward the back, the left shoulder, and the loins, and it is likely to recur. Fever as high as 102° often accompanies the pain; and there may be nausea, vomiting, and diarrhœa. The attack resembles hepatic colic, and the colic is a stage in which the disease has progressed considerably without being recognized. The spleen will then be found enlarged and tender, and it may have irregularities on its surface, especially during the painful seizures.

Rarely there is some perisplenitis, and this may be accompanied by pleurisy at the left base. There may be respiratory trouble, irregular reflex cough, some subcrepitant rales, and a diminution of the vesicular breathing sound. The perisplenitis seems to be a mechanical effect of the splenic growth.

The anæmia begins as a simple anæmia in the slow cases. Then there is a decrease of the erythrocytes and of hæmoglobin. There may be some poikilocytosis, no considerable change in the leukocytes, or a slight increase of the multinuclear neutrophiles and the blood plates.

Where there is a tendency to hemorrhage, the hæmoglobin is commonly proportionately lower than the number of red corpuscles; the multinuclears decrease, the lymphocyts remain near the normal ratio, the eosinophiles may be above the normal number, and more or less nucleated red cells may appear, but never so many as in pernicious anæmia.

Hæmatemesis is frequently though not constantly found in splenic anæmia. It is of splenic origin rather than hepatic, and it is very lasting. Osler said, "With the exception of the chronic hemorrhagic form of peptic ulcer there is no known condition in which hæmatemesis may occur for so many years." Recurrent epistaxis and bleeding from the gums may occur, and in a few cases hemorrhages into the eye fundus have been noticed. Hæmaturia and hæmoptysis are very rare.

In general, after the spleen is enlarged, the anæmia, which may last from three to ten or more years, is a marked secondary anæmia, but it has no specific characteristics, except that ordinarily in splenic anæmia the ratio of the hæmoglobin to the number of erythrocytes is lower than in other secondary anæmias. Microcytes are frequently observed. Usually there is leukopenia, but after hemorrhage, or from some intercurrent inflammatory process, there may be some leukocytosis. No micro-organism has been found in the blood.

Commonly the enlarged spleen is smooth and it retains its customary outline; indeed, any marked deviation from the outline makes the diagnosis doubtful. It may extend in beyond the navel and down to the iliac crest. The enlargement is permanent in nearly every case. If there is a cessation of this growth, the spleen does not recede in a typical case. Strümpell reported a case where the size of the spleen diminished for a while under treatment.

The liver dulness frequently goes a half-inch or more below the normal line, and jaundice is found, though rarely. Petechiæ, especially on the legs, show in the advanced stage.

The external lymphatic glands do not swell, or at most one or two glands are merely palpable. In autopsies, however, the mesenteric glands are commonly found enlarged. Bruhl would make this condition of the external glands a differential diagnostic sign in splenic anæmia.

In the cachectic stage there may be œdema, ascites, or hydrothorax. Asthenia is progressive, and diarrhœa and hemorrhages are more frequent. There is in this stage frequently considerable fever of a hectic type, which may reach 103° or 104° in the evening. The lesions of splenic anæmia are seen only in the spleen and blood, and perhaps secondarily in the liver. Other organs are affected exceptionally and indirectly.

The disease is fatal, and the treatment that has proved of value up to the present is either splenectomy before the cachectic stage, or Talma's operation. Banti advocates splenectomy in all conditions except (1) in profound cachexia; (2) in pseudoleukæmic hypertrophy of the lymphatic glands. He thinks ascites indicates cachexia. Colzi, however, according to Banti, did a splenectomy in a case in which there was hepatic cirrhosis with some hob-nailed growth, and the patient recovered. Jean Roger[1] reported two splenectomies with recovery, each done in the ascitic stage of Banti's disease. Bessel-Hagen recommends splenectomy in any stage of the disease. Schiassi[2] reported a case of Banti's disease in the third stage, where he fixed the spleen to the parietal peritoneum. This cured the ascites, but the anæmia persisted. Twelve to fifteen grams of fresh bone-marrow from the epiphyses, and one to three grams of hepatic extract were given to the patient, and the anæmia disappeared.

Jordan,[3] of Heidelberg, collected 17 splenectomies for splenic anæmia, with 14 recoveries, a mortality of 17.65 per cent. Harris and Herzog[4] had already collected 19 cases with 14 recoveries—a mortality of 26.32 per cent. We found 13 additional splenectomies for splenic anæmia done since 1901 (there may have been a considerable number more than that) in which there were ten recoveries and three deaths. If we add these to Harris and Herzog's list there will be 32 cases with twenty-four recoveries—a mortality of 25 per cent. Without splenectomy or Talma's operation all die; with splenectomy about 75 per cent. recover; the statistics for Talma's operation as applied to splenic anæmia are not extensive enough to warrant any conclusion.

The patient that was the source of this present article was a white boy, aged eighteen years. He had no history of syphilis, alcoholism, or malaria, and he was under observation from June 16, 1904, to the time of his death, November 30, 1904. When first seen his fat was about normal, his skin was very sallow generally, and the scleras somewhat jaundiced. Along the median line of the belly the superficial veins were varicose. The spleen was smooth, and so enlarged that it went down to the crest of the ilium, and inward to a finger's breadth beyond the navel, and its outline was normal. The liver was normal apparently. The urine was high in color, somewhat overacid, with an excess of alkaline phosphates, but otherwise it was normal.

During the preceding April he had had severe pain in the region of the spleen, with nausea and vomiting, and he had had nose-bleed "since childhood." In July, 1903, he had had remittent fever (Widal reaction negative), diarrhœa with some blood in it, bleeding from the gums and nose. Dr. Samuel Wolfe, who saw him in the Samaritan Hospital at that time, says "the spleen then extended over to the right of the umbilicus." When first seen in June, 1904, quinine and arsenic were prescribed.

Between June 17 and July 16 the spleen contracted 5 cm. in length, and the body weight increased from 98 to 112 pounds. The spleen remained unchanged in size for some time, but toward the end of October it had contracted another 5 cm.—10 cm. in all (about 4 inches). The body weight increased, with one loss of two pounds toward the end of September, from 98 pounds on June 17 to 124 pounds on October 22; shortly after this time ascites set in.

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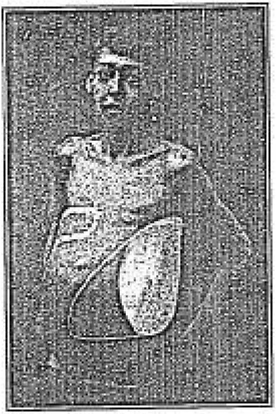
[1] La presse médicale, No. 59, vol. x.

[2] Gazz. degli ospedali, 1902, No. 69.

[3] Berliner klin. Woch., December 28, 1903.

[4] Annals of Surgery, July, 1901.

There was no hæmatemesis during the course of the disease. The gums bled somewhat almost nightly; nose-bleeding occurred only twice after June 17. The temperature went up as high as 100° or 101° frequently, and on October 27 there was dyspnœa and a temperature of 105°. The patient had one severe attack of diarrhœa and another lighter than this.



The white space is the splenic dulness on November 5, 1904; the narrow white line is the dulness on June 16, 1904. The broad white line is the liver-dulness on November 5, 1904.

The urine showed no signs of nephritis until November 21, after the ascites had appeared. There was no albumin at any time (potassium ferrocyanide test) until after the splenectomy, when a faint cloud was precipitated twice. On November 21 a few medium-sized hyaline casts and some with leukocytes appeared. Twice these casts were so numerous as to be remarkable in the absence of albumin.

At the beginning of October the left inguinal glands, the right postcervical, and some in the anterior cervical triangle were enlarged enough to be palpable. This slight swelling disappeared from the inguinal glands before the end of the month, but one of the post-cervical glands remained palpable. The liver dulness remained normal until the beginning of November, when it showed signs of contraction.

The skin was always very sallow. At the beginning of November petechiæ showed on the legs, and the scrotum became pigmented very darkly—like an argyria stain. On November 5th minute hemorrhagic spots were observed in both optic disks, but none elsewhere in the fundi. The retinal veins were twice the width of the arteries, which were normal.

The blood examinations were as follows:

July 10*th*. Red corpuscles, 4,350,000; hæmoglobin, 80 per cent. (three observers); color index, 1.08; some poikilocytosis; many unstained centres. At this and every succeeding blood examination the blood was searched for the malaria plasmodia, with a negative result.

16*th*. Reds, 2,450,000; whites, 14,062; hæmoglobin, 85 per cent.; color index, 1.73; the intercorpuscular spaces took the stain deeply. A new Tallqvist scale was used, but three observers agreed upon the hæmoglobin percentage.

August 6*th*. Reds, 3,000,000; whites, 14,000; hæmoglobin, 80 per cent.; color index, 1.33; the intercorpuscular spaces took the stain deeply. Two observers.

October 15*th*. Differential count (whites, 8800):

Polymorphonuclear neutrophiles	57.5
Small lymphocytes	28.5
Large lymphocytes	6.5
Eosinophiles	0.5
Myelocytes	1.5
Basophiles	1.5
Large mononuclears	4.0

November 5*th*. Reds, 1,630,000; whites, 5400; hæmoglobin, 65 per cent. (a Tallqvist scale, three observers); color index, 1.99. The reds were very pale, many were broken down, and they took the stain feebly; the intercorpuscular spaces took the stain. The blood coagulated so slowly it was difficult to stop it with collodion.

15*th*. Reds, 1,930,000; whites, 2800; hæmoglobin, 75 per cent.; color index, 1.94. This count is an average of four counts, repeated to avoid possible error; and the percentage of hæmoglobin was agreed to by four observers. The interspaces stained.

21*st*. Reds, 3,130,000; whites, 8800; hæmoglobin, 65 per cent.; color index, 1.03; specific gravity, 1.050; Dare hæmoglobinometer, three observers. Interspaces stained.

22*d*. Reds, 3,090,000; whites, 3800; hæmoglobin, 77 per cent.; color index, 1.24. Interspaces stained. The blood coagulated in two and a half minutes.

23*d*. Splenectomy.

24*th*. Reds, 2,770,000; whites, 6400; hæmoglobin, 69 per cent.; color index, 1.24.

28th. Reds, 3,150,000; whites, 9600; hæmoglobin, 68 per cent.; color index, 1.07. Six normoblasts were found in four slides (not cover-glasses).

A specimen of the serum, obtained upon the day the splenectomy was performed, was sent to Dr. W. M. L. Coplin, professor of pathology in the Jefferson Medical College, and the diluted serum, free, of course, from cells, gave the characteristic narrow alpha-band between D and E, marginating D; and a wider further beta-band, also between D and E. Dr. Coplin said the serum contained hæmoglobin, and more than its color indicated.

A blood examination made on May 3, 1904, before the patient came under our observation, was: reds, 2,410,000; whites, 2659; hæmoglobin, 75 per cent.; color index, 1.55. This high color index agrees with our finding.

The necessity of splenectomy had been explained to the family of the patient early in the summer, but they would not permit the operation. When he had become ascitic and was suffering from the consequent pressure, they asked that splenectomy be done. The condition of cachexia was a strong contraindication, but the success of Roger and Colzi in three cases in a similar state, and the opinion of Bessel-Hagen, were taken as a justification for doing splenectomy as a last chance.

Dr. William J. Taylor, visiting surgeon to St. Agnes' Hospital, removed the spleen. The incision through the belly wall was made from the border of the ribs to below the crest of the ilium along the outer border of the rectus muscle, and a second incision at right angles to this from the main incision outward toward the flank. There were a few slight adhesions to the diaphragm, but much adhesion along the lower border of the spleen to the sigmoid flexure of the colon. The mesenteric attachment of the spleen was stripped, the vessels dissected loose and tied with silk. One friable vein broke, but the bleeding was easily controlled. All adhesions and attachments were ligated and cut and the spleen lifted out of the belly. It weighed 945 grams. When oozing had been stopped, a strip of iodoform gauze was passed down to the stumps of the vessels for packing and drainage. The ascitic fluid welling in the belly caused considerable delay by covering the field of operation. Dr. Taylor says that in another similar case he would drain the belly by making a small opening and turning the patient on his side before operating.

The day after the operation the patient, who had reacted well after the splenectomy, became restless, and the temperature went up to 102.3°. The next day he was stuporous, his pupils dilated, and the head was slightly retracted; three days later there was some rigidity of the muscles in general, and twitching of the left facial muscles. Injections of salt solution restored consciousness, but he relapsed into the stuporous condition. The temperature ranged from 99.3° to 102°, and there were involuntary voidings of urine and feces. The operation wound became septic with the colon bacillus from his own handling of the dressing. He died a week after the splenectomy.

The Autopsy. Pathological Diagnosis: (chronic splenic tumor); atrophic hepatic cirrhosis; septic peritonitis; pancreatitis; parenchymatous nephritis; slight cardiac fibrosis; pigmentation of the skin.

The body was emaciated; the skin was very sallow; the scrotum was of a blackish hue; there were petechiæ on each patella. The cœliotomy wound was purulent.

The belly: the subcutaneous fat was absent; the bowels were very anæmic and covered with purulent and flocculent exudate, and the bottom of the pelvic cavity was filled with pus. This pus, as was learned afterward, was caused by an infection from the bacillus coli communis through the cœliotomy wound. The omentum was congested and thickened throughout, and newly adherent to the stomach and over its whole surface to the belly wall. It was friable at the gastric adhesions.

The liver was firmly adherent to the diaphragm and the transverse colon, and newly adherent to the omentum. The liver was gray, hob-nailed, dense, atrophied. It weighed 910 grams. The gall-bladder was normal.

The appendix vermiformis was bound down by old adhesions. The stomach was very anæmic, inflamed in the region of the abdominal wound, and it was newly adherent to the diaphragm and the belly wall. The pancreas was indurated and very firmly adherent to all the organs that touched it, so that it was difficult to get it out. The adrenal bodies were normal. The kidney capsules stripped with some resistance, and on section the kidneys were slightly congested. The mesenteric glands were slightly enlarged.

Nothing unusual was found in the thorax except old adhesions at the back of the left lung. The skull was not opened through lack of permission.

Microscopic Appearances. The spleen. There was a connective-tissue hyperplasia throughout the organ. The splenic substance was transformed by this thickening and a marked proliferation of endothelium. All the bloodvessels and sinuses had thickened walls from endothelial proliferation, and these cells were so packed into the sinuses as to fill them completely throughout the greater part of the spleen. Many of the Malpighian bodies were recognizable, but they were enlarged three or four times by endothelial proliferation, and were irregular in outline. There were giant cells with nuclei grouped centrally and peripherally, as in Stengel's third case,[1] but these cells were not so numerous as they were in his case. A search for tubercle bacilli was negative.

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[1] Loc. cit.

The liver had the ordinary microscopic evidences of atrophic cirrhosis. No unusual endothelial proliferation or metastasis could be found.

The pancreas showed interstitial inflammation and a considerable atrophy of tissue. There was a slight interstitial fibrosis in the heart muscle, and considerable epithelial degeneration in the kidneys, especially in the convoluted tubules, and a moderate congestion everywhere. The adrenal bodies and the lungs were normal.

The striking characteristics of this case are: (1) the fact that the spleen shrank 10 cm. during our observation—Strumpell's is the only similar case we find; (2) there was a marked true hæmoglobinæmia throughout the last six or seven months of the patient's life. A characteristic of this disease is a low color index from a decrease in the hæmoglobin; in this case the corpuscles were laked out, but the hæmoglobin seemed to remain in the serum.

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HYPERNEPHROMA: REPORT OF THREE CASES.

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Until embryologists decide where to classify a certain new-growth found in the kidney the non-commital term hypernephroma is a happy one. Whether these tumors are to be placed with the sarcomas, carcinomas or adenomas is one of the debatable questions